
Magdeburg
11/9-12/9
ABINEP
Symposium
Cormack
Sherrington
Edelman
Watson
Edward
Wiesel
Hounsfield
Pavlov
Fechner
Mendel
Bordet
Pasteur
Metchnikoff
Edinger
Behring
Erikson
Broca
Berger
Crick
Wundt
Landsteiner
Adler
Hubel
Porter
Bell
Galen
Ehlrich
Meyer
Freud
Ogawa
Skinner
Cajal
Jenner
Weber
Willis
Westphal
Müller
Varolio
Schwann
Ranvier
Alzheimer
Wernicke
Sylvius
Penfield
Brodmann
Purkyně
Helmholtz

1st Annual Symposium of the International Graduate School ABINEP

September 11th - 12th, 2018

Keynote Speakers

Dr. Mike X **Cohen** (Nijmegen, Netherlands)

Dr. Sonja **Djudjaj** (Aachen, Germany)

Prof. Helmut **Kettenmann** (Berlin, Germany)

Prof. Ole **Paulsen** (Cambridge, UK)

Lukasklause, Magdeburg

Germany

CONTACT

Dr. Christiane Hedtmann
Project Coordinator
OVGU ESF ABINEP
International Graduate School on Analysis, Imaging and Modeling of Neuronal and Inflammatory Processes
Otto-von-Guericke University Magdeburg, Medical Faculty, Institute of Physiology, H13/222,Leipziger Str. 44, 39120, Magdeburg.

ORGANIZERS

Alexander Pausder, Aneri Shah, Ann-Kathrin Meinshausen, Ayse Malci, Babak Khodaie
Babak Saber Marouf, Beatrice Barbazzeni
Camila Agostino, Carla Marcia Cangalara Lira
Dr. Christiane Hedtmann, Ehsan Kakaei
Evangelia Pollali, Isabel Bernal
Lisa Osbelt, Julia Rogge
Rituparna Bhattacharjee, Sarah Schreier
Sharavanan Ganesan, Stefan Repplinger
Timothy French, Vivekanandhan Viswanathan, Prof. Volkmar Lessmann and Yuan Wang

Local Organizer

ABINEP Students

The international Graduate school (GS) on Analysis, Imaging, and Modeling of Neuronal and Inflammatory Processes (**ABINEP**) is based on the two internationally recognized biomedical research foci of the Otto-von-Guericke-University Magdeburg (OVGU), Neurosciences and Immunology, ABINEP aims at fostering cutting edge research projects in rising subdisciplines of these research areas, which are currently supported by several German Research foundation (DFG)- and European Community (EU)-funded collaborative projects in Magdeburg including the DFG-funded Collaborative Research Centers SFBs 779 and 854 and associated graduate schools, as well as DFG TRRs 31 and 62.
The program includes scientists from the **Medical Faculty/ University Hospital Magdeburg (FME)** and the **Faculty of Natural Sciences (FNW)** of the OVGU, the **Institute for Neurobiology (LIN)** and **German Center for Neurodegenerative Diseases (DZNE)**, both located in Magdeburg, the **Helmholtz Centre of Infection Research** in Braunschweig as well as international collaborators.

Sponsors

Special thanks to

Jens Strackeljan, Monika Brunner-Weinzierl, Volkmar Lessmann, Constanze Seidenbecher, Christiane Hedtmann and Anke Ryll
for administrative support.

All the ABINEP Students for contributing
to organize this annual symposium.

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Foreword

Welcome to the 2018 ABINEP Student Symposium, the annual symposium for graduate students from the International Graduate School on Analysis, Imaging and Modeling of Neuronal and Inflammatory Processes.

The ABINEP Symposium provides graduate students with the opportunity to present their research and network with like-minded colleagues in a professional environment. This experience is invaluable for young graduate students, many of whom will continue on to present at national and international conferences. In addition, the ABINEP Symposium allows graduate students from across Magdeburg to learn about exciting research being conducted by their peers, whether from different institutes or from across different disciplines. We hope that the knowledge gained from this symposium will aid in stimulating new research ideas and establishing future collaborations.

The ABINEP Symposium will connect brain researchers from across Magdeburg research institutes and will include oral presentations and posters spanning four unique themes comprised of: inflammatory processes in neurodegeneration (Module 1), neurophysiology and computational modeling of neuronal networks (Module 2), infection and immunity in the context of aging (Module 3), and human brain imaging for diagnosing neurodegenerative disorders (Module 4). Attendees are also invited to a poster session with ABINEP students to highlight the outstanding presentations and projects. This would give us an opportunity for a networking session where we all

can discuss the exciting research we've been exposed to throughout the course of these two days (over a cup of coffee, of course)!
Thank you for attending our ABINEP Student Symposium, we encourage you to actively connect with your fellow research students within and outside of your discipline.

Keynote Speakers

Prof. Helmut Kettenmann (Berlin, Germany) – *Guest speaker, Module 1*

The Role of Microglial Cells in Brain Diseases

Prof. Helmut Kettenmann and his group aim to understand the role of glial cells in physiology and pathology. This includes the sensing of neuronal activity by astrocytes, the communication between astrocytes and their feedback on neurons. They also focus on the expression of transmitter receptors in microglial cells and how the microglial function is influenced by the activation of these receptors. Furthermore, they investigate the interaction between glioma cells and intrinsic brain cells with the goal of understanding these processes on a molecular level.

Prof. Ole Paulsen (Cambridge, UK) - *Guest speaker, Module 2*

Neuromodulation of Hippocampal Synaptic Plasticity: Mechanisms and Possible Behavioural Implications

Prof. Ole Paulsen and his team focus on the relationship between network oscillations and synaptic plasticity. They combine different techniques (whole-cell patch-clamp and planar multi-electrode recording, calcium imaging and light-activated channels) to investigate how information can be stored and retrieved as changes of synaptic weights in neural networks displaying oscillatory activity.

Dr. Sonja Djudjaj (Aachen, Germany) - *Guest speaker, Module 3*

Role of Macrophage Inhibitory Factor (MIF) in Renal Diseases.

Dr. Sonja Djudjaj and her group focus on research in the field of nephropathology. Currently, she is working on renal inflammation and fibrosis and in particular the role of the Macrophage Inhibitory Factor (MIF).

Dr. Mike X Cohen (Nijmegen, Netherlands) – *Guest speaker, Module 4*

Midfrontal Theta: A Burgeoning Multiscale Perspective

Dr. Mike X Cohen and his team focus on multiscale electrophysiology, incorporating single-unit, LFP, and EEG recordings. Dr. Cohen also develops online teaching materials for learning programming, signal processing, statistics and big-data analysis.

ABINEP Institutes

BMMR = Biomedizinische Magnetresonanz
DZNE = Deutsches Zentrum für Neurodegenerative Erkrankungen
FME = Medizinische Fakultät
FNW = Fakultät für Naturwissenschaften
HZI = Helmholtz-Zentrum für Infektionsforschung
IBIO = Institut für Biologie
IBZ = Institute für Biochemie und Zellbiologie
IEIM = Institute für Experimental Internal Medicine
IEP = Institute of Experimental Physics
IfP = Institute of Psychology
IHG = Institut für Humangenetik
IIN = Institut für Inflammation und Neurodegeneration
IKND = Institute für Kognitive Neurologie und Demenzforschung
IMMB = Institut für Medizinische Mikrobiologie und Krankenhaushygiene
IMKI = Institut für Molekulare und Klinische Immunologie
IPA = Institute für Pathologie
IPHY = Institute für Physiologie
IPSY = Institut für Psychologie
IPT = Institute für Pharmakologie und Toxikologie
LIN = Leibniz-Institut für Neurobiologie
KGHI = Universität für Gastroenterologie, Hepatologie und Infektiologie
KHAE = Universitätsklinik für Hematologie und Onkologie
KHNO = Universitätsklinik für Hals-Nasen-Ohrenheilkunde, Kopf-und Halschirurgie
KNEU = Universität für Neurologie

KNEP = Universitätsklinik für Nieren -und Hochdruckkrankheiten, Diabetologie und Endokrinologie
KOBI = Kognition Biology Group at IBIO
KORT = Orthopädische Universitätsklinik
KPNE = Universitätsklinik für Pneumologie
OVGU = Otto-von-Guericke Universität
UKMD = Universitätsklinikum Magdeburg

ABINEP Fellows

Module 1: Neuroinflammation: Inflammatory Processes in Neurodegeneration

(Students Names are in *Italics* and Supervisor(s) Names are in **Bold**)

1) Ms. Sarah Schreier

Prof. Dr. rer. nat. Daniela Dieterich (OVGU, FME, IPT)

Prof. Dr. rer. nat. Andrea Kröger (OVGU, FME, IMMB)

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2) *Mr. Timothy French*

Prof. Dr. rer. nat. Ildiko Dunay (OVGU, FME, IIN)

PD Dr. rer. nat. Björn Schott (LIN)

---

3) Ms. Ayse Malci

Prof. Dr. rer. nat. Eckart Gundelfinger (LIN)

Prof. Dr. rer. nat Michael Naumann (OVGU, FME, IEIM)

Prof. Dr. rer. nat. Constanze Seidenbecher (LIN)

---

4) Ms. Rituparna Bhattacharjee

PD Dr. rer. nat. Eike Budinger (LIN)

PD Dr. med. Jürgen Goldschmidt (LIN)

Dr. rer. nat. Xu Wang (OVGU, FME, IMMB)

Dr. rer. nat. Nishanth Gopala (OVGU, FME, IMMB)

Prof. Dr. med. Dirk Schlüter (OVGU, FME, IMMB)

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5) Ms. Carla Marcia Cangalaya Lira

Prof. Dr. rer. nat. Klaus-Dieter Fischer (OVGU, FME, IBZ)

Prof. Dr., Ph.D. Alexander Dityatev (DZNE)

Dr., Ph.D. Stoyan Stoyanov (DZNE)

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Module 2: Neurophysiology and Computational Modeling of Neuronal Networks

(Students Names are in *Italics* and Supervisor(s) Names are in **Bold**)

1) Mr. Babak Khodaie

Prof. Dr. rer. nat. Volkmar Lessmann (OVGU, FME, IPHY)

Dr. rer. nat. Elke Edelmann (OVGU, FME, IPHY)

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2) Mr. Ehsan Kakaei

Prof. Dr., Ph.D. Jochen Braun (OVGU, FNW, IBIO)

Prof. Dr. rer. nat. Oliver Speck (OVGU, FNW, BMMR)

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3) Ms. Evangelia Pollali

Prof. Dr. sc. nat. Oliver Stork (OVGU, FNW, IBIO)

Dr. rer. nat. Thomas Munsch (OVGU, FME, IPHY)

4) Mr. Vivekanandhan Viswanathan

Prof. Dr. rer. nat. Frank Ohl (OVGU, FNW, IBIO, LIN)

Dr. rer. nat. Andreas Schulz (LIN)

----

5) Mr. Babak Saber Marouf

Dr., Ph.D. Motoharu Yoshida (DZNE, LIN)

Prof. Dr., Ph.D. Magdalena Sauvage (LIN)

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Module 3: Immunosenescence: Infection and Immunity in the Context of Aging

(Students Names are in *Italics* and Supervisor(s) Names are in **Bold**)

1) Ms. Lisa Osbelt

Prof. Dr. med. Thomas Fischer (OVGU, FME, KHAE)

Prof. Dr. med. Dirk Schlüter (OVGU, FME, IMMB)

PD Dr. Till Strowig (HZI)

OA Dr. med. Enrico Schalk (OVGU, FME, KHAE)

OÄ Dr. med. Jacqueline Färber (OVGU, FME, IMMB)

Prof. Dr. med. Martin Zenker (OVGU, FME, IHG)

---

2) Ms. Ann-Kathrin Meinshausen

Prof. Dr. Andreas Müller (OVGU, FME, IMKI)

Prof. Dr. rer. nat. Jessica Bertrand (OVGU, FME, KORT)

Prof. Dr. med. Christoph Lohmann (OVGU, FME, KORT)

Prof. Dr. rer. nat. Dietmar Pieper (HZI)

Prof. Dr., Ph.D. Eva Medina (HZI)

3) Mr. Alexander Pausder

Prof. Dr. rer. nat. Dunja Bruder (OVGU, FME, IMMB, HZI)

Prof. Dr. med. Jens Schreiber (OVGU, FME, KPNE)

Dr. rer. nat. Julia Boehme (OVGU, FME, IMMB, HZI)

PD Dr. Till Strowig (HZI)

---

4) Ms. Aneri Tusharbhai Shah

Prof. Dr. rer. nat. Ingo Schmitz (OVGU, FME, IMKI, HZI)

Prof. Dr. med. Peter Mertens (OVGU, FME, KNEP)

Dr. rer. nat. Xenia Gorny (OVGU, FME, KNEP)

Prof. Dr. rer. nat. Dunja Bruder (OVGU, FME, IMMB, HZI)

---

5) Ms. Isabel Bernal

Prof. Dr. rer. nat. Dunja Bruder (OVGU, FME, IMMB, HZI)

Prof. Dr. med. Ali Canbay (OVGU, FME, KGHI)

Prof. Dr. rer. nat. Lothar Jänsch (HZI)

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6) Ms. Yuan Wang

Prof. Dr. med. Christoph Arens (OVGU, FME, KHNO)

Prof. Dr. rer. nat. Michael Naumann (OVGU, FME, IEIM)

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Module 4: Human Brain Imaging for Diagnosing Neurocognitive Disorders

(Students Names are in *Italics* and Supervisor(s) Names are in **Bold**)

1) Ms. Beatrice Barbazzeni

Prof. Dr. med. Emrah Düzel (OVGU, FME, IKND, DZNE)

Prof. Dr. rer. nat. Oliver Speck (OVGU, FNW, BMMR)

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2) Mr. Sharavanan Ganesan

Prof. Dr. phil. Stefan Pollmann (OVGU, FNW, IPSY)

Jun.-Prof. Dr. rer. nat. Michael Hanke (OVGU, FNW, IPSY)

---

3) Ms. Camila Silveira Agostino

Prof. Dr. rer. nat. Tömme Noesselt (OVGU, FNW, IPSY)

Prof. Dr.-Ing. Hermann Hinrichs (OVGU, FME, KNEU)

---

4) Ms. Julia Rogge

Prof. Dr. med. Markus Ullsperger (OVGU, FNW, IPSY)

PD Dr. rer. nat. Gerhard Jocham (OVGU, FWW, CNL)

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5) Mr. Stefan Repplinger

Dr. phil. Tino Zähle (OVGU, FME, KNEU)

Prof. Dr. med. Hans-Jochen Heinze (OVGU, FME, KNEU)

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General Information

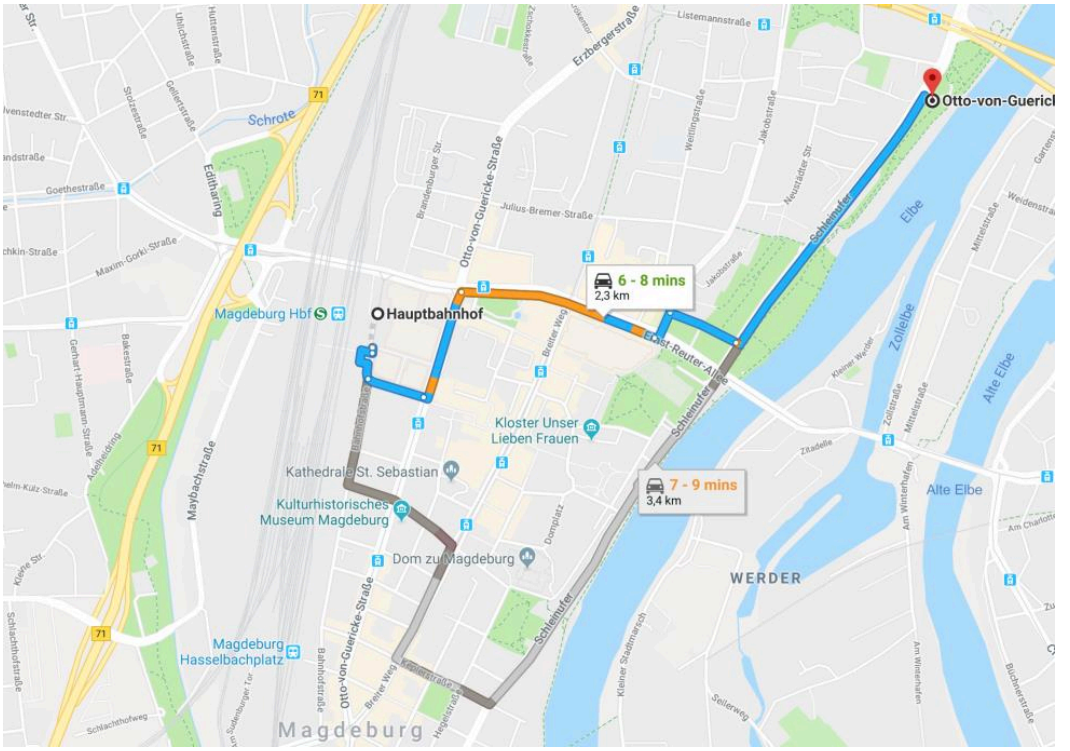
Lukasklause

Key Information

Way to the conference center

Address

Lukasklause
Otto-von-Guericke Museum
Schleinufer 1
39104 Magdeburg



Since 1996, the Lukasklause has held various cultural events for the public. The focus is on Otto von Guericke, the history of the Old City of Magdeburg in the 17th century, as well as other topics of his time for the Magdeburg region, which are of significance to our time

1. Internet Access

A Wireless LAN environment is available in the conference center. The WiFi login details will be provided at the day of the conference.

2. Lunch:

Snacks and coffee will be provided at the conference centre during the breaks. Alternatively, you can eat in one of the suggested Restaurant (see "Recreational Information" section of the booklet).

3. Students/Poster Session and Round Table

Each day from 12:35 to 13:35 takes place a "student/poster session" (in the foyer room of Lukasklause) and a "round table" at the end of each day with PhD students and Keynote Speakers only. Students have the opportunity to present their research in a more conversational context, to learn about exciting research being conducted by their peers from across different disciplines, to get feedback from the "Keynote Speakers" and stimulating new research ideas. We encourage students to take the advantage of this unique opportunity to ask the questions they never "dared" to.

4. Dinner

On the first day of ABINEP Symposium there will be a BBQ at 17:30 at the Lukasklause. This event would give us the opportunity to interact in a more informal context, sharing research ideas, ABINEP Symposium considerations and maybe building future research collaborations. We hope you will enjoy this pleasant "networking session" (over a glass of wine and tasty food).

5. Hotel Motel One

Motel One Magdeburg is a stylish combination of science and design. It is located directly next to Magdeburg Cathedral and the state parliament, making it the perfect base for a tour of Otto von Guericke's city. Magdeburg central station is within easy reach of the hotel and offers convenient connections to Leipzig-Halle and Berlin airports.

Address

Motel One Magdeburg

Domplatz 5

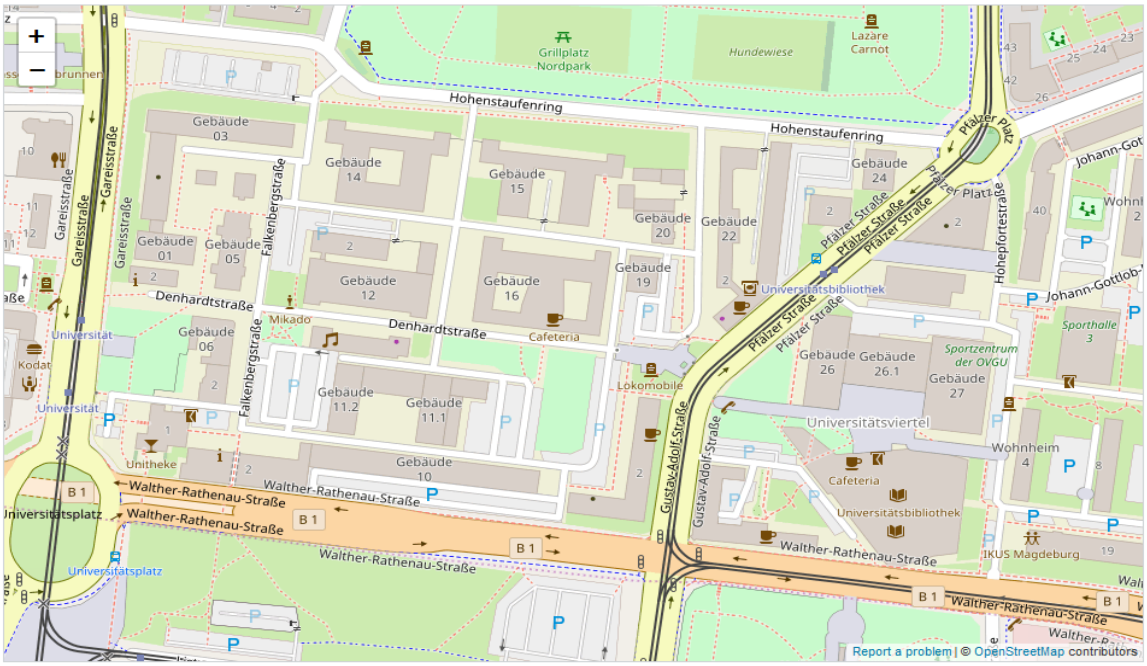
39104 Magdeburg

By car:

On both days of this Symposium, ABINEP students will take the responsibility to pick Keynote Speakers up from the Motel One to the location of the ABINEP Symposium by car.

Map of the University

University Campus



Medical Campus

Program

ABINEP Symposium 2018
of the **International Graduate School ABINEP**

Sep 11th-12th, 2018 | Lukasklause, Magdeburg, GER

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Program September 11, 2018

09:00–09:10	**Welcome by Prof. Dr. Volkmar Leßmann**
Session I:	**Module 4-** Human Brain Imaging for Diagnosing Neurocognitive Disorders *Chairs: Julia Rogge, Alexander Pausder*
09:10–10:20	*Guest speaker-* **Dr. Mike X Cohen** (Nijmegen, Netherlands) *Midfrontal Theta: A Burgeoning Multiscale Perspective*
10:20-10:45	**Coffee Break I**
10:50–11:05	**Beatrice Barbazzeni** *Cognitive Training Based on EEG-Neurofeedback to Improve Working Memory in Preclinical Alzheimer's Disease*
11:05–11:20	**Julia Rogge** *Mechanisms of Evidence Accumulation during Perpetual Decision Making*
11:20–11:35	**Camila Agostino** *No Effect of Temporal Information Manipulation on Statistical Learning: a pilot study*
11:35-12:35	**Lunch Break**
12:35–13:35	**Poster Session**

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Program September 11, 2018

Session II:	**Module 1-** Neuroinflammation: Inflammatory processes in Neurodegeneration *Chairs: Ayse Malci, Camila Agostino*
13:35–14:45	*Guest speaker-* **Prof. Helmut Kettenmann** (Berlin, Germany) *The Role of Microglial Cells in Brain Diseases*
14:45–15:00	**Rituparna Bhattacharjee** *Development of New Techniques for Visualisation of Neuroinflammatory Processes during Infections and Autoimmunity Diseases of the Brain- Neuroinflammation in Experimental Cerebral Malaria*
15:00-15:25	**Coffee Break II**
15:30–15:45	**Carla Marcia Cangalara Lira** *Cytoskeleton-Dependent Mechanisms of the Microglia-Matrix-Neuron-Interaction During Neuroinflammatory Processes*
15:45–16:00	**Ayse Malci** *Neuroplastin Signaling and Synaptic Plasticity in Normal and Neuroinflammatory Conditions*
16:00–17:00	**'PhD students-Guest speakers only' round table** (no PIs)
17:00	**BBQ + Get together**

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Program September 12, 2018

09:00 – 09:10	**Welcome**
Session III:	**Module 3-** Immunosenescence: Infection and Immunity in the Context of Aging *Chairs: Alexander Pausder, Aneri Shah*
09:10–10:20	*Guest speaker-* **Dr. Sonja Djudjaj** (Aachen, Germany) *Role of Macrophage Inhibitory Factor (MIF) in Renal Diseases*
10:20-10:45	**Coffee Break I**
10:50–11:05	**Lisa Osbelt** *Influence of the Intestinal Microbiome on Infections, Course Disease and Success of Treatment on Cytostatic Drug-Treated Hemic-Oncological Patients*
11:05–11:20	**Alexander Pausder** *Elucidating the Roles of Secretory Immunoglobulins in Asthma under Homeostatic and Infectious Conditions*
11:20–11:35	**Isabel Bernal** *Characterisation of Innate Antibacterial T-cell Immunity to Understand Age-Associated Infections with C. difficile*
11:35-12:35	**Lunch Break + Photograph session**
12:35–13:35	**Poster Session**

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Program September 12, 2018

Session IV:	**Module 2-** Neurophysiology and Computational Modelling of Neuronal Networks *Chairs: Evangelia Pollali, Vivekanandhan Viswanathan*
13:35–14:45	*Guest speaker-* **Prof. Ole Paulsen** (Cambridge, United Kingdom) *Neuromodulation of Hippocampal Synaptic Plasticity: Mechanisms and Possible Behavioral Implications*
14:45–15:00	**Babak Khodaie** *Distinct Properties of Spike timimg-dependent LTP at Schaffer collateral-CA1 Synapses along the Dorso-Ventral Axis of the Mouse Hippocampus*
15:00-15:25	**Coffee Break II**
15:30–15:45	**Ehsan Kakaei** *Modelling of Dopamine-Induced Neuronal Network Activity - "Learning Conditional Associations: Rich Temporal Context and Involvement of Hippocampus / Medial Temporal Lobe"*
15:45–16:00	**Babak Saber Marouf** *Simulation of Behaviour-Dependent Network Activity and Dynamics on the Basis of In Vivo and In Vitro Recording*
16:00–16:15	**Evangelia Pollali** *Modulation of Behaviour-Related Oscillations by Interneuron Networks*
16:15–17:15	**'PhD students-Guest speakers only' round table** (no PIs)
17:15 – 17:30	**Closing remarks**

Poster Session - Students

Module 1
Neuroinflammation: Inflammatory Processes in Neurodegeneration

Sarah Schreier
Reaction of Brain-Resident Cell Types During Neurotropic Virus Infection

Timothy French
Infection-induced Neuroinflammation is altered by an Intestinal Nematode Infection

Carla Marcia Cangalara Lira
Cytoskeleton-Dependent Mechanisms of the Microglia-Matrix-Neuron-Interaction During Neuroinflammatory Processes

Module 2
Neurophysiology and Computational Modelling of Neuronal Networks

Babak Khodaie
Distinct Properties of Spike timimg-dependent LTP at Schaffer collateral-CA1 Synapses along the Dorso-Ventral Axis of the Mouse Hippocampus

Vivekanandhan Viswanathan
Dopamine-Dependent Modulation of Neuronal Circuits in the Auditory Cortex and the Striatum

Ehsan Kakaei
Modelling of Dopamine-Induced Neuronal Network Activity - "Learning Conditional Associations: Rich Temporal Context and Involvement of Hippocampus / Medial Temporal Lobe"

Evangelia Pollali
Enhanced hippocampal network oscillations in a mouse model of reduced GABA synthesis

Poster Session - Students

Module 3
Immunosenescence: Infection and Immunity in the Context of Aging

Aneri Shah
Role of Y-box Binding Protein 1 in Activation of NF-κB signalling pathway Downstream

Ann-Kathrin Meinshausen
Interleukin-8 and the Terminal Complement Pathway Identify Posthesis Infection in Periprosthetic Tissue Samples

Alexander Pausder
Expression of the Murine Polymeric Immunoglobulin Receptor (pIgR) under Homeostatic and Immune Modulating Conditions

Yuan Wang
Metagenomic Survey of Microbiota Function in Laryngeal Tissues in the Development of Dysplasia and Carcinoma

Module 4
Human Brain Imaging for Diagnosing Neurocognitive Disorders

Beatrice Barbazzeni
Cognitive Training Based on EEG-Neurofeedback to Improve Working Memory in Preclinical Alzheimer's Disease

Julia Rogge
Mechanisms of Evidence Accumulation during Perpetual Decision Making

Sharavanan Ganesan
Visual Search and Attention Guidance in Patients with Macular Degeneration

Stefan Repplinger
Effort processing in the substantia nigra – intracranial recordings from patients suffering from Parkinson's disease

Camila Agostino
No Effect of Temporal Information Manipulation on Statistical Learning: a pilot study

Keynote Speaker Talks

Midfrontal Theta: A Burgeoning Multiscale Perspective

Cohen M X

Radbound University, Donders Centre for Neuroscience, Nijmegen, Netherlands

Midfrontal theta is an idiosyncratic electrophysiological signature of that feeling when you are just about to make a mistke. I'll give a lightning-fast overview of the key findings in this literature and then discuss the current state-of-the-art, which includes (1) our demonstrations that conflict-related midfrontal theta is non-phase-locked and unrelated to ERPs, (2) our struggles to understand why, despite being 100% non-phase-locked, midfrontal theta phase seems to be so important for connectivity and for behavior, and (3) the curious invisibility of conflict-related theta in MEG. Several of the biggest mysteries are off-limits in humans, and I'll close by explaining how our current/future work in acquiring multiscale ephys datasets (neurons, LFP, EEG) in rodents will help make new discoveries about the origins and significances of midfrontal theta.

The role of microglial cells in brain diseases

Kettenmann H

Max Delbreuck Center for Molecular Medicine in the Helmholtz Association, Berlin, Germany

Ten years ago we promoted the concept that microglial activation is not an all or none process but is highly diverse depending on the type of pathology and time point during the pathologic process. We have, therefore, studied aspects of microglial properties in mouse models of Alzheimer's Disease, schizophrenia, and glioma. In Alzheimer's Disease two functions of microglial cells are impaired, namely the phagocytic activity and the ability to respond to a local injury. Phagocytic activity is controlled by P2Y6 receptors and we recently found that also purinergic signaling is impaired. An impairment of phagocytic activity was also found in microglia isolated from a mouse model of schizophrenia. In glioma, microglial and invading monocytes accumulate and these glioma associated brain macrophages (GAMs) phagocytic activity is increased. The GAM phenotype is altered in a very characteristic manner not reflecting the classical M1 or M2 phenotype of activation. We found two mechanisms altered in GAMs which helped to promote glioma growth, namely the upregulation of metalloproteases MT1/MMP and MMP9. This supports the hypothesis that microglial cells can obtain diverse phenotypes depending on the pathologic state.

Role of MIF in renal diseases

Djudjaj S

Institute of Pathology - University Hospital RWTH, Aechen, Germany

Macrophage migration inhibitory factor (MIF) is a cytokine best known for its proinflammatory function in a number of diseases including atherosclerosis, autoimmune diseases and sepsis. In this study the role of MIF was analyzed in the context of different renal diseases.
In patients with glomerulonephritides, MIF and its receptor CD74 were highly upregulated. Analyses of a murine model of immune-mediated glomerulonephritis showed that MIF aggravates the disease by promoting the pathological proliferation of glomerular cells via its receptor CD74.
The common final pathway of virtually all progressive renal diseases is chronic kidney disease (CKD) with interstitial fibrosis as a major hallmark. We hypothesized that MIF might be involved in fibrosis progression by its proinflammatory function. Surprisingly, we found a decreased MIF expression in fibrotic tissue. Analyses of a large set of animal and in vitro experiments showed that in the CKD, MIF had an unexpected anti-fibrotic and anti-inflammatory role. Here MIF also enhanced the cell proliferation and reduced the cell-cycle arrest leading to an increased tubular epithelial cell regeneration. Finally in the setting of acute kidney injury we have shown using a combination of clinical studies, animal models and in vitro experiments, that MIF is also renoprotective.

Taken together, MIF has a dual role in kidney diseases, promoting (auto)immune glomerular diseases and limiting tubular cell injury in the setting of acute and chronic kidney diseases.

Neuromodulation of hippocampal synaptic plasticity: Mechanisms and possible behavioural implications

Paulsen O

Behavioural and Clinical Neuroscience Institute, Department of Physiology, Development and Neuroscience, University of Cambridge, Cambridge, UK.

An important challenge in contemporary neuroscience is to bridge between events at cellular and behavioural levels. Whereas hippocampal synaptic plasticity is widely accepted as a cellular model of memory, it has been difficult to reconcile the time scale of induction of synaptic plasticity with the time scale of behavioural events. In particular, it is difficult to explain how conventional synaptic plasticity could associate behaviour with delayed outcomes of that behaviour. We propose that neuromodulation of synaptic plasticity may provide a link to close this explanatory gap.
Spike timing-dependent plasticity (STDP) is a physiologically realistic form of Hebbian learning which is found in the hippocampus. In its classic form, STDP depends on the order and precise timing of presynaptic and postsynaptic spikes at a millisecond time scale: pre-before-post spike pairings induce timing-dependent long-term potentiation, whereas post-before-pre pairings induce timing-dependent long-term depression. However, these timing requirements are profoundly influenced by neuromodulators, including acetylcholine and dopamine. In the rodent hippocampus, acetylcholine is released during exploratory behaviour, whereas dopamine sig-

nals novelty and/or reward. Using whole-cell recording in hippocampal slices, we found that acetylcholine biases STDP towards depression, whereas dopamine biases STDP towards potentiation and can even convert depression into potentiation when applied after the plasticity protocol, a phenomenon referred to as 'retroactive modulation'. Incorporating this bidirectional neuromodulated synaptic learning rule into a computational model of mouse foraging behaviour, we find effective navigation toward changing reward locations, as in natural foraging behaviour. The predictions from the computational model were confirmed using optogenetics during natural mouse behaviour. Thus, temporally sequenced neuromodulation of STDP enables associations to be made between actions and outcomes and provides a possible mechanism for aligning the time scales of cellular and behavioural learning.

ABINEP Fellows

Talks and Posters

☑ ***Both poster and oral presentations***
☐ ***Only poster presentation***

Cognitive Training Based on EEG-Neurofeedback to Improve Working Memory in Preclinical Alzheimer's Disease ☑

Barbazzeni B[1,3,4,8], Düzel E[1,2,3,4,8], Speck O[1,2,5,6,7,8,9,10]

[1]ESF International Graduate School on Analysis, Imaging and Modelling of Neuronal and Inflammatory Processes; [2]German Center for Neurodegenerative Diseases; [3]Faculty of Medicine; [4]Institute for Cognitive Neurology and Dementia Research; [5]Faculty of Natural Sciences; [6] Department of Biomedical Magnetic Resonance; [7]Institute for Physics; [8]Otto-von-Guericke University of Magdeburg, Germany; [9]Leibniz Institute for Neurobiology, Magdeburg, Germany; [10]Center for Behavioral Brain Sciences, Magdeburg, Germany.

Memory impairments in Alzheimer's disease (AD) have been associated with progressive hippocampal atrophy, impaired brain oscillatory activity, decreased attention and motivation to learn while encoding new stimuli. Although neuropharmacological interventions have been the major approach to treat cognitive impairments, none of them have shown convincing efficacy in stopping the progression of cognitive decline and AD pathology. Recently, the combination of cognitive training (CT) with neurofeedback (NF) has demonstrated to enhance memory and brain oscillatory activity, particularly when applied during the early stage of the pathology (preclinical AD, pAD). Similarly, the anticipation of reward in a monetary-reward-learning situation has been correlated with alpha suppression increase; the

neural correlate of attentional and memory processes. Thus, to improve working memory (WM) in pAD (older adults who underwent a previous Amyloid PET/MRI imaging evaluation), the current research project aims to combine CT with EEG-neurofeedback (EEG-NF) to train alpha suppression during the performance of a monetary-rewarded delayed match-to-sample task. The study will evaluate the effect of monetary-reward incentives and CT combined with EEG-NF training on WM, as well as, on hippocampal volume changes by implementing high-field magnetic resonance imaging (MRI). Furthermore, a three months follow-up will evaluate whether EEG-NF training of alpha suppression facilitates transfer effects on a different cognitive task. The goal of the current research is to develop new methods as a novel and personalized therapeutic tool to aid pAD patients and to ameliorate up-regulation of brain oscillatory activity, attention and motivation to learn, consequently improving memory and life quality.

Mechanisms of Evidence Accumulation during Perceptual Decision Making ☑

Rogge J[1,2], Jocham G [1,3], Ullsperger M[1,2]

[1]ESF International Graduate School on Analysis, Imaging and Modelling of Neuronal and Inflammatory Processes; [2]Institute of Psychology, Otto-von-Guericke University of Magdeburg, Germany; [3]Cognitive Neuroscience, Center for Behavioral Brain Sciences, Otto-von-Guericke University of Magdeburg, Germany

Perceptual decision making as an essential part of every cognitive process requires the accumulation of evidence for a stimulus among several other steps of information processing. In two-alternative forced-choice tasks, the effect of evidence strength on performance can be successfully modelled by a drift-diffusion model (Ratcliff, 2008). Neural correlates of evidence accumulation potentially involve more than the centro-parietal positivity (CPP) (Kelly & O'Connell, 2013). In tasks requiring two-sided effectors, such as left- or right-handed button presses, sensorimotor areas show lateralization effects. Specifically in the beta-band, motor areas show pronounced suppression contralateral to the effector side prior to response execution (Donner et al., 2009). This beta-lateralization possibly not only encodes motor preparation but also the accumulation of sensory evidence. With a random-dot motion paradigm, this study aims at showing the relevance of beta-lateralization in evidence processing. Electroencephalography was recorded in thirty healthy young human participants to investigate the temporal dynamics of the beta-lateralisation and modelling will be used to relate the signal to latent computational variables. Initial behavioural analyses confirm a behavioural effect of evidence strength on performance parameters (reaction time and accuracy) with less noisy stimuli leading to better performance. With this experiment we aim at building a foundation for further investigations on the influence of urgency on perceptual decision making and neurochemical substrates of perceptual decision making.

No Effect of Temporal Information Manipulation on Statistical Learning: a pilot study ☑

Agostino S C[1,2], Ball F[2,], Hinrichs H[3], Noesselt T[2]

[1]ESF International Graduate School on Analysis, Imaging and Modelling of Neuronal and Inflammatory Processes; [2]Insistute of Psychology, Otto-von-Guericke University of Magdeburg, Germany, [3]Department of Neurology, Medicine Faculty Otto-von-Guericke University of Magdeburg

Investigations in statistical learning have suggested that individuals tend to readjust their predictions to new statistical environment over time. However, it remains unclear whether the manipulation of temporal information may affect the learning of new regularities. To test the hypothesis that the temporal information has an effect on human prediction accuracy and learning rates, nineteen volunteers (five women, mean age 28.15) took part in the experiment. The task consisted of a checkerboard object that moved between three positions using Markov Chains. The probabilities for each trajectory resulted in different movement patterns (e.g. circle or semicircle). After the object completed the movement, it disappeared and participants indicated where they expected the ball to go next. The number of trials per pattern was based on individual learning rates. Temporal information was manipulated by manipulating movement velocity (constant velocity, probability dependent velocity and random velocity) and shape of movement patterns (circular, semicircular and elliptical). We analyzed learning rate and prediction accuracy, using repeated measures ANOVAs. Results indicated that the manipulation of temporal information did not affect performance (F(2,28) =

0.43, p =0.654; F(2,28) = 0.126, p = 0.882, respectively). In contrast, there was a modulation of learning rate/prediction accuracy by shape of patterns (F(2,28) = 8, p = 0.002; accuracy by shape: F(2,28) = 10.6, p < 0.001, respectively). These preliminary results do not support the hypothesis that temporal information affects learning rate and prediction accuracy, however, the spatial pattern of new regularities may play an important role during acquisition and prediction.

Development of New Techniques for Visualization of Neuroinflammatory Processes of the Brain – Neuroinflammation in Experimental Cerebral Malaria ☑

Bhattacharjee R[1,2], Arnim S[2], Budinger E[3], Oelschlegel A[3], Goldschmidt J[3], Matuschewski K[4,5], Schlüter D[2,6], Nishanth G[2]

[1]ESF International Graduate School on Analysis, Imaging and Modelling of Neuronal and Inflammatory Processes;[2]Institute of Medical Microbiology and Hospital Hygiene, Otto-von-Guericke University, Magdeburg, Germany; [3]Leibniz Institute for Neurobiology, Magdeburg, Germany; [4]Parasitology Unit, Max Planck Institute for Infection Biology, Berlin, Germany; [5]Department of Molecular Parasitology, Humboldt University, Berlin, Germany; [6]Helmholtz Centre for Infection Research, Braunschweig, Germany

Cerebral malaria is the major complication of severe malaria and may result in the death of infected individuals. The mouse model of experimental cerebral

malaria (ECM), is widely used to study the pathogenesis of human cerebral malaria. To visualise the progression of asymptomatic malaria *in vivo*, we used single-photon emission computed tomography (SPECT) technique. C57BL/6 mice were injected with *Plasmodium berghei* ANKA-infected red blood cells (RBCs). On day 5 post infection (p.i.), one group of mice was treated with the anti-malarial drug pyrimethamine, while the control group received phosphate buffered saline (PBS). SPECT imaging showed diffused hypo-perfusion in the olfactory bulbs and cortical regions of PBS treated mice early at day 5 p.i., much before the onset of ECM symptoms, which progressed until day 7 p.i. Pyrimethamine treatment reduced vascular dysfunction resulting only in mild hypo-perfusion in the cortical areas at day 7 p.i. Furthermore, the parasite load in the brain of pyrimethamine treated mice reduced significantly until day 7 p.i. and the mice did not develop symptoms of ECM. These data indicate that, similar to human malaria, intracerebral accumulation of parasitised RBCs is essential for the development of ECM. In addition, pyrimethamine treatment reduced brain pathology, including blood brain barrier disruption, brain haemorrhage, neuroinflammation, astrogliosis, infiltration of pathogenic $CD8^+$ T cells and apoptosis of endothelial cells. These changes were visible only on day 7 p.i., particularly in the olfactory bulb and brain stem regions. The novel finding of our study is that pyrimethamine treatment protected the rostral migratory stream (RMS) integrity and fostered neurogenesis, while the RMS was completely disrupted in the PBS treated mice explaining why cerebral malaria resulted in *impaired cognitive functions.* Taken together, our study delineates the pathophysiological

alterations in the brain during the progression of ECM, thereby providing a better understanding of the disease pathogenesis.

Cytoskeleton-dependent mechanisms of the microglia-extracellular matrix-neuron interaction during neuroinflammatory processes ☑

Cangalaya C[1,2,3], Stoyanov S[3], Gottfried A[2,4], Fischer K D[2,4], Dityatev A[3,4,5]

[1]ESF International Graduate School on Analysis, Imaging and Modelling of Neuronal and Inflammatory Processes, [2]Institute of Biochemistry and Cell Biology, [3]German Center for Neurodegenerative Diseases, Molecular Neuroplasticity Group, Magdeburg, [4]Otto-von-Guericke University of Magdeburg, Medical Faculty, Germany, [5]Center for Behavioral Brain Sciences, Magdeburg, Germany

Neuroinflammation is a common feature in dementias. Microglia functions and their ability to survey the neuropil can be impaired during neuroinflammation. Cytoskeleton dysfunction and overexpression of small GTPases in microglia, as well as upregulation in the extracellular matrix (ECM), have been reported in neurodegenerative diseases and dementia. Moreover, there is evidence that the enzymatic digestion of ECM, antibodies against specific ECM glycoepitopes and the treatment with inhibitors of small GTPases can restore the synaptic transmission/plasticity in dementia/neuroinflammatory conditions. Thus, the perisynaptic ECM and cytoskele-

ton-dependent mechanisms are likely to play a crucial role in the interaction between microglia and synapses. In order to investigate these phenomena, we aim to study how ECM components and actin cytoskeleton regulate microglia-neurons interactions during inflammation. We will perform time-lapse studies of microglia interactions with neurons and ECM, and the effects of this interaction on synapse formation/elimination under normal physiological conditions and in neuroinflammation. Moreover, we will investigate the role of small GTPases and actin cytoskeleton-regulated processes in microglia interactions with neurons and ECM. To perform these studies, we are using conditional knockout models of cytoskeletal regulators, and chondroitinase ABC injection/knockdown of specific ECM components for ECM attenuation. Adeno-associated viruses expressing fluorescently tagged HAPLN1, Thy1-GFP and Cre-reporter mice are employed for in vivo labeling of ECM, dendritic spines, and microglia, respectively. Microglia activation and interaction with synapses in vivo are studied using a single cell/spine photo-ablation with two-photon microscopy. Phagocytic functions of microglia are also evaluated by in vivo injection of fluorescently labeled A□42 and ex vivo analysis of intramicroglial localization of A□42/synaptic markers in fixed tissue by confocal microscopy and flow cytometry-based assays.

Neuroplastin signaling and synaptic plasticity in normal and neuroinflammatory conditions ☑

Malci A[1,2,3], Naumann M[4], Gundelfinger ED[3,5], Seidenbecher CI[3,5], Herrera-Molina R[3]

[1]*ESF International Graduate School on Analysis, Imaging and Modelling of Neuronal and Inflammatory Processes* [2]*Otto-von-Guericke-University Magdeburg, Faculty of Natural Sciences;* [3]*Leibniz Institute for Neurobiology, Department of Neurochemistry and Molecular Biology Magdeburg, Germany;* [4]*Institute of Experimental Internal Medicine, Otto-von-Guericke-University Magdeburg, Germany;* [5]*Center for Behavioral Brain Sciences (CBBS) Magdeburg, Germany*

Neuroplastins (Nps) are type-1 transmembrane proteins with extracellular Ig-like domains important for the regulation of synapse plasticity and formation/stabilization, the balance of excitatory/inhibitory synapse ratio, and for the homeostasis of cytosolic calcium. It is proposed that *via trans*- and *cis*-binding, neuroplastins could form multimers with transmembrane and cytosolic proteins to modulate cell signaling pathways involved in the before mentioned processes. We know that Nps regulate ERK and NFκB activation during the development of neurons. Currently, we are characterizing how Np can also regulate ERK during plasticity in electrically stimulated mature neurons. Interestingly, in mature neurons and T cells, neuroplastins form complexes with the plasma membrane calcium ATPase (PMCA) to regulate calcium homeostasis. As calcium-dependent signaling cascades and gene transcription are known to be essential mechanisms involved in synapse plasticity, we propose that the dysfunction of Np-PMCA complexes during pathophysiologically relevant conditions such as neuroinflammation should result in impaired plastic capacity of neurons. Therefore, we hypothesize that neuroinflammatory conditions may alter the normal function of Np-

PMCA mediated calcium clearance, and in turn, impair downstream signaling cascades (i.e. ERK or NFκB). To test this hypothesis, we monitor ERK activation by combining Western blot and immunostaining with biosensor-based live cell imaging in hippocampal neurons. In future, neuroplastin-PMCA driven signaling cascades will be studied under pathophysiological conditions by checking a potential relevance of the complex dysfunction in neuroinflammation models.

Reaction of brain-resident cell types during neurotropic virus infection ☐

Schreier S[1,2,6], Zegenhagen L[3], Jänsch L[3], Dieterich D C[4,5,6], Kröger A[2,3,6]

[1]ESF International Graduate School on Analysis, Imaging and Modelling of Neuronal and Inflammatory Processes, [2]Institute of Medical Microbiology and Hospital Hygiene, [3]Helmholtz Centre for Infection Research, [4]Institute of Pharmacology and Toxicology, [5]Leibniz Institute for Neurobiology, [6]Otto-von-Guericke University of Magdeburg, Germany

Tick-borne encephalitis virus (TBEV) is an important neurotropic human pathogen in Europe and Asia. Although TBEV infections of the nervous system are rare, the consequences are often severe. These include encephalitis and meningoencephalitis, which has a fatality rate of 0.5-2.0% for Central European

strains and 20-40% for Far Eastern strains. No antiviral drug is currently available [Yellow Book, CDC, 2018]. Although the factors controlling viral neuroinvasiveness and neuro-pathogenicity are poorly defined, the innate immune response, especially the type I Interferon (IFN) response, is essential for survival.

Previous work has demonstrated that the type I IFN response from neuroectodermal cells is important for survival of LGTV infection in mice [Weber et.al 2014]. Moreover, type I IFN induction in distinct brain regions depends on various factors of the type I IFN system [Weber et. al 2014; Kurhade et. al 2016]. In particular, the induction and expression of IFN-β in astrocytes is regulated by a different, interferon regulatory factor 7 (IRF7) independent mechanism, in the brain [Zegenhagen, *unpublished*]. Variable protein expression in the astrocytes of different brain regions might play an important role in antiviral signaling.

Using cell-specific proteome labeling, I will investigate how astrocytes react to the infection, change their activation and function during the course of viral infection. Additionally I will investigate their impact on the inflammatory response in the brain.

So far I investigated an over-expression of the intermediate filament glial fibrillary acidic protein (GFAP), an indicator for reactive astrocytes, in the Olfactory bulb in IRF7$^{-/-}$ and mitochondrial antiviral signaling (MAVS) protein knock out mice after viral infection.

Influence of the intestinal microbiome on infections, course of disease and success of treatment on cytostatic drug-treated hemic-oncological patients ☑

Osbelt L [1,2,3,4,5], Färber J [1,2,5], Schalk E [1,3,5], Schlüter D [1,2,5], Thomas F [1,3,5], Strowig T [1,4]

[1]ESF International Graduate School on Analysis, Imaging and Modelling of Neuronal and Inflammatory Processes,[2]Institute for Medical Microbial Microbiology and Hygiene,[3]Clinic for Oncology and Hematology, [4]Microbial Immune Regulation, Helmholtz Centre Infection Research, Braunschweig, [5]Otto-von-Guericke University of Magdeburg, Germany

Infectious diseases caused by *Clostridium difficile* and multidrug-resistant gram negative bacteria (MRGN) are a major problem during the therapy of hematological-oncological patients associated with prolonged hospital stays and increasing mortality rates (1). Especially patients suffering from acute myeloid leukemia (AML) and patients receiving blood-cell or bone-marrow transplantation are highly susceptible towards infections and development of graft-versus-host diseases (GvHD) (2). The microbiota is a complex community of bacteria, archaea, fungi and eukaryotes colonizing all body surfaces and plays a fundamental role regarding the resistance against invading pathogens and the maintenance of the barrier function in healthy humans. There is evidence that the development and course of mucositis, colitis and GvHD is also associated with changes in the microbiome of the patients (3-5). During this project we aim to investigate the role of the

microbiome regarding the susceptibility to infections, the course of disease and the persistence of MRGN and *Clostridium difficile* in cytostatic-treated mice and AML patients.
To address these questions, we characterized the microbiome composition before, during and after chemotherapy in mice and humans. We demonstrated that different isogenic C57BL/6N mouse lines respond with dramatic changes in the microbiome composition which follow a fluctuating pattern along the course of therapy. Even though similar changes could be observed in different isogenic mouse lines with regard to the bacterial families which are decreased or increased during chemotherapy, some mouse lines seem to be more prone for development of intestinal mucositis. In future, we would like to address the question whether and how microbial changes affect susceptibility to inflammation and infection during chemotherapeutically induced dysbiosis and would like to correlate our findings in mice with our data from a human cohort of AML patients.

Effect of rich temporal context on visual object recognition studied by novel 3D objects ☑

Kakaei E[1,2,3], Aleshin S[2,3], Speck O[3,4,5,6], Braun J[2,3]

[1] ESF graduate school ABINEP, Otto-von-Guericke University, Magdeburg, Germany; [2] Institute of Biology, Otto-von-Guericke University, Magdeburg, Germany; [3] Center for

Behavioral Brain Sciences, Magdeburg, Germany;[4] Biomedical Magnetic Resonance, Otto-von-Guericke University, Magdeburg, Germany [5] German Center for Neurodegenerative Disease, Magdeburg, Germany;[6] Leibniz Institute for Neurobiology, Magdeburg, Germany.

Temporal context is believed to boost the visual object recognition. To investigate this effect further, we suggest a novel way of generating complex 3D objects by tensor multiplication of Bezier curves (https://goo.gl/qACZaJ). This method allows generating various families of objects with desired similarity and dissimilarity -within and between families- where the similarity is calculated as the Euclidean distance between the 3 orthogonal projections of the objects.
Observers showed improved performance in identifying the different objects and learned to distinguish between familiar objects and novel objects presented in a sequence of objects composed of 90% of objects from six repeating families and 10% of objects from novel families, observers rapidly learn to distinguish between familiar and novel objects, as well as between familiar and novel families of objects. In addition, observers showed rapid recognition and slower sequence learning after being exposed to temporal context created by Hamiltonian sequences of 15 families of objects.
Moreover, for the future studies and to prepare for fMRI data analysis, a MATLAB-based toolbox (https://git.io/fNAA0) have been developed to facilitate application of the novel functional sub-parcellation method provided by *Dornas, J. V., & Braun, J. (2018)*.

Enhanced hippocampal network oscillations in a mouse model of reduced GABA synthesis ☑

Pollali E[1,2,3,4], Çalışkan G[2], Munsch T[1,3,4], Lessmann V[1,3,4], Stork O[1,2,4]

[1]ESF International Graduate School on Analysis, Imaging and Modelling of Neuronal and Inflammatory Processes, [2]Department of Genetics and Molecular Neurobiology, Institute of Biology, [3] Institute of Physiology, Medical Faculty,[4] Otto-von-Guericke University of Magdeburg, Germany

GAD65 KO mice, a model of reduced γ-aminobutyric acid (GABA) production, exhibit fear- and anxiety-phenotype. At the same time, network oscillations, that are dependent on inhibition and the neurotransmitter GABA, have been linked to memory processes, such as spatial memory formation and also in vitro, in ventral hippocampus (vHP) are modulated by stress-reactive factors. Therefore, by characterizing the network oscillations in GAD65 KO mice, the aim of this study is to find out a potential mechanism of how interneuron dysfunction leads to stress phenotype and a potential involvement of oscillations in emotional memories.
We performed local field potential (LFP) recordings from acute horizontal brain slices including the vHP and recorded the spontaneous Sharp-Wave Ripples (SW-R) and carbachol-induced gamma oscillations from Cornu Ammonis 1 (CA1) and 3 (CA3). We found that the power and the peak frequency of gamma oscillations were significantly increased both in CA3

and CA1 subregions of GAD65 KO mice in comparison to wild-type (WT). In line, spontaneous SW-R activity was augmented in the CA3, with increased amplitude of both SW- and Ripple- components of the oscillation.
These findings suggest that reduced GABA availability in GAD65 KO mice may trigger long-term alterations in the vHP network oscillations, which may underlie the fear- and anxiety- phenotype of GAD65 KO mice. Thus, augmented network oscillations in the vHP might be a risk factor for the development and persistence of fear memories and stress-related disorders.

Distinct Properties of Spike timing-dependent LTP at Schaffer collateral-CA1 Synapses along the Dorso-Ventral Axis of the Mouse Hippocampus ☑

Khodaie B[1,3], Edelmann E[1,2,3] and Lessmann V[1,2,3]

[1]Institute of Physiology, Otto-von-Guericke University Magdeburg, [2]Center for Behavioral Brain Sciences (CBBS), Magdeburg, [3]OVGU ESF-founded International Graduate School ABINEP 39120 Magdeburg, Germany.

The processing of memory information is mainly governed by the hippocampus in association with some cortical and subcortical regions. Based on the functional, behavioral, anatomical, and gene expression properties, the hippocampus can be divided into three separate compartments: Dorsal (DH), intermediate (IH), and ventral hippocampus (VH). Moreover, CA1 pyramidal cells (CA1 PCs) are also heterogeneous along the dorso-ventral axis with respect to their neuromodulatory and excitatory input and output fibers. Currently, little is known about the differences in activity-dependent plasticity measured in single postsynaptic CA1 PCs. Using spike timing-dependent plasticity (STDP) recorded with whole cell patch clamp technique this study evaluates t-LTP magnitude in identified CA1 PCs along the longitudinal axis of hippocampus. Recordings were performed in acute hippocampal slices from P28-P36 C57Bl6/J mice in CA1 PCs. Two STDP protocols, 1) a canonical (1 pre and 1 postsynaptic stimulation with 6 repeats- 6x 1:1, and 2) a burst protocol (6x 1:4) were used to induce t-LTP. We also applied burst STDP protocols with low number of repeats (3x) and longer time intervals (spike timing interval, 40 and

100 ms) between pre- and postsynaptic stimulations. Our results revealed robust t-LTP induction with both STDP paradigms in Schaffer collateral-CA1 synapses along the dorso-ventral axis. Results demonstrate a higher magnitude of potentiation in DH CA1 PCs using both STDP protocols compared to IH and VH CA1 PCs. Furthermore, the burst STDP protocol induced a higher magnitude of t-LTP compared to the canonical protocol. We also observed robust t-LTP in response to a burst protocol with 3x 1:4 stimulation. Unexpectedly, this 3x 1:4 t-LTP seems to be controlled less stringently by exact spike timings. Together these data proposed that the synaptic potentiation along the longitudinal axis of the hippocampus might be correlated with the diversity of their afferent inputs, physiological synaptic and intrinsic properties.

Dopamine-dependent modulation of neuronal circuits in the auditory cortex and striatum ☐

Viswanathan V[1,2,3], Lippert M T[2,3], Schulz A L[1,2], Ohl F[1,2,3]

[1]ESF International Graduate School on Analysis, Imaging and Modelling of Neuronal and Inflammatory Processes; [2]Leibniz Institute for Neurobiology; [3]Otto-von-Guericke University of Magdeburg, Germany

Acoustic information about the world is processed in the auditory system. This sensory system is connected to a host of other structures in the brain such as other cortices, neuromodulatory nuclei and motor centers, necessary to meaningfully interpret sensory

cues and guide behavior accordingly. One of the structures of particular importance for decision making and execution is the striatum. Via a proposed 'direct' and 'indirect' pathway, the striatum influences action selection and execution in response to arriving auditory—or other sensory—information. Since in nature behavioral contingencies change over time, these processes cannot be static but have to be plastic for the animal to survive. Exactly how this plasticity of striatal representations in response to auditory information and changing reward contingencies manifests is unknown.

A major experimental limitation to investigate this question is the heterogeneity of striatal neurons, composed of both dopamine D1 and D2-receptor carrying neurons with highly dissimilar functions. Neurons carrying the D1-receptor are associated with the direct pathway, a neuronal circuit facilitating movement. In contrast, those carrying the D2-receptor belong to the indirect pathway, suppressing movement. With traditional methods, such as unit recordings, these cells cannot reliably be distinguished. In my thesis I therefore investigate the role of direct and indirect pathway neurons in the auditory striatum during auditory discrimination learning using optogenetic activity indicators. These indicators allow me to selectively label and optically image striatal cells with genetic specificity. For example, I can specifically target my imaging to only D1 or D2-positive neurons, and selectively image their activity. In parallel to imaging, mice are trained in a novel spatial auditory discrimination task including conditions of contingency reversal. I expect to observe a differential change in activity of D1 versus

D2-positive neurons with regard to stimulation, action and reward consummation in the process of learning and reversal.

Role of Y-box binding protein 1 in activation of NF-κB signaling pathway downstream ☐

Shah A[1,2,3,6,7], Gorny X[1,2,7], Lindquist JA[1,2,7], Bruder D[1,5,6,7], Schmitz I[1,3,6,7], Mertens PR[1,2,7]

[1]ESF International Graduate School on Analysis, Imaging and Modelling of Neuronal and Inflammatory processes; [2]Clinic of Nephrology and Hypertension, Diabetes and Endocrinology; [3]Institute of Molecular and Clinical Immunology; [4]Research Group of Systems-Oriented Immunology and Inflammation Research, [5]Institute of Medical Microbiology and Hospital Hygiene; [6]Helmholtz Centre for Infection; [7]Otto-von-Guericke University of Magdeburg, Germany

The immune system constitutes the first line of defense averse to invading pathogens. Thereby, phagocytic cells like monocyte/macrophage combat and clear pathogens via secretion of cytokines like tumor necrosis factor (TNF), IL-1, IL-6, IL-8 and IL-12. Immune cell recruitment into affected tissue is a hallmark of autoimmune diseases, such as lupus erythematodes, sarcoidosis, membranous glomerulopathy, IgA nephritis and rheumatoid arthritis. The cold-shock Y-box binding protein YB-1 is important for monocyte/macrophage differentiation and phagocytic function. It is already known that monocyte

differentiation is impaired when cold shock YB-1 protein is genetically depleted in conditional *Ybx1* knockout mice with loss of phagocytic activity. Mechanism of phagocytosis also involves activation of intracellular signaling cascades, amongst others leading to the activation of NF-κB. Similarly, it is demonstrated that the NF-κB modulator $I\kappa B_{NS}$ regulates IL-10 expression and macrophages lacking IκB proteins exhibit altered differentiation into classical, pro-inflammatory M1 and alternatively activated M2 macrophages.

To test for the crosstalk between YB-1 and NF-κB signaling in the orchestration of macrophage functions, lentiviral transduced YB-1 knockdown monocytic THP-1 cells were generated, which showed a prominent role of YB-1 in the phosphorylation of the NF-κB subunit p65. Imaging flow cytometry indicated reduced nuclear translocation of NF-κB p65 in YB-1 knockdown cells after TNF-α stimulation compared to non-transfected i.e. wildtype cells. Expression of TRAF (Tumor necrosis receptor-associated factor) 2 , which is a prerequisite for activating NF-κB upon TNF-α stimulation, was significantly reduced in the absence of YB-1. This suggests a role of YB-1 in the recruitment of TRAF2 and thereby activation of NF-κB downstream signaling pathway.

Infection-induced neuroinflammation is altered by an intestinal nematode infection ☐

French T[1,2], Schött B[1,3], Dunay I[1,2]

[1]ESF International Graduate School on Analysis, Imaging and Modelling of Neuronal and Inflammatory Processes; [2]Institute for Inflammation and Neurodegeneration, Otto von Guericke University Magdeburg, Germany; [3]Leibniz Institute for Neurobiology, Magdeburg, Germany

Given the high incidence of helminth and protozoan infection in developing countries, it is very likely that a vast number of humans are co-infected. Thus there is growing interest in understanding how an existing infection affects the capacity of the host to develop a proper response to subsequent infections. *Toxoplasma gondii* (*T. gondii*) infection elicits a strong Th1 immune response characterized by increased levels of IFNγ, IL-12, iNOS and TNF. Previously the reactivated toxoplasmosis in immunocompromised individuals has received considerable attention, and recently became evident that the latent infection is associated with basal neuroinflammation responsible for neuronal and behavioral alterations of the host. A helminth infection, such as *Heligmosomoides polygyrus* (*H. polygyrus*), elicits a strong Th2 type immune response associated with increased levels of IL-4, IL-5 and IL-13. Helminths have evolved strategies for survival that can either suppress the immune response or subvert it to an ineffective response. The effects of helminth infection on peripheral inflammation are well studied, however little is known on the alterations caused by co-infections in the central nervous system. Therefore, we aimed to

investigate the effect of intestinal helminth infection on the development of *T. gondii* infection-induced neuroinflammation in a murine model.

Expression of the murine polymeric immunoglobulin receptor (pIgR) under homeostatic and immune modulating conditions ☐

Pausder A[1,2,3], Boehme J[1,2,3], Strowig T[1,4], Schreiber J[1,5], Bruder D[1,2,3]

[1]ESF International Graduate School on Analysis, Imaging and Modelling of Neuronal and Inflammatory Processes; [2]Institute of Medical Microbiology and Hospital Hygiene, Otto-von-Guericke-University Magdeburg, Germany; [3]Immune Regulation Group, Helmholtz Centre for Infection Research Braunschweig, Germany; [4]Junior Research Group Microbial Immune Regulation, Helmholtz Centre for Infection Research Braunschweig, Germany; [5]Clinic for Pneumology, Otto-von-Guericke-University Magdeburg, Germany

The transport of secretory immunoglobulin A and M trough the epithelial cell barrier into the mucosal lumen by the polymeric immunoglobulin receptor (pIgR) represents an important mechanism in terms of mucosal host defence in the respiratory tract. Currently, there is only limited information on how *Pigr* gene expression is affected by immune modulating stimuli in the lung and the upper respiratory tract. It is important to identify such stimuli because they might be applicable in a therapeutic way.

The goal of this project is to elucidate *Pigr* gene expression under homeostatic and immune modulating conditions in areas of the respiratory tract of mice, which are crucial for the mucosal host defence. *Pigr* gene expression in the lung, trachea and nasal-associated lymphoid tissue (NALT) of naïve, LPS-treated and germ-free mice and mice with a chronic autoimmune-mediated lung inflammation was determined by quantitative real-time PCR.
Pigr gene expression gradually decreases from the upper to the lower respiratory tract. Increased *Pigr* gene expression in the lung of LPS stimulated mice and mice with a chronic autoimmune-mediated lung inflammation were detected. Germ free mice showed no significant differences in the *Pigr* expression pattern compared to conventional specific-pathogen-free mice.
These data suggest that *Pigr* gene expression is site-specific and can be modulated by microbial ligands and microbial-independent inflammatory processes. Furthermore, it was shown that changes in the expression of *Pigr* are not a reaction to microbial colonization but an intrinsic effect.

Clostridioides difficile activates human mucosal-associated invariant T cells ☑

Bernal I[1,2,3], Hofmann J D[4], Bulitta B[1], Klawonn F[3,5], Michel A[6], Jahn D[6], Neumann-Schaal M[4,7], Bruder D[2,8], Jänsch L[3]

[1]ESF International Graduate School on Analysis, Imaging and Modelling of Neuronal and Inflammatory Processes; [2]Institute of Medical Microbiology and Hospital Hygiene, Infection Immunology, Otto von-Guericke University Magdeburg; [3]Structure and Function of Proteins, Helmholtz Centre for Infection Research, Braunschweig; [4]Braunschweig Integrated Centre of Systems Biology (BRICS), Department of Bioinformatics and Biochemistry, Technical University Braunschweig; [5]Department of Computer Science, Ostfalia University of Applied Sciences, Wolfenbüttel; [6]Braunschweig Integrated Centre of Systems Biology (BRICS), Department of Microbiology, Technical University Braunschweig; [7]Leibniz Institute DSMZ-German Collection of Microorganisms and Cell Cultures, Braunschweig; [8]Immune Regulation, Helmholtz Centre for Infection Research, Braunschweig, Germany

Clostridioides difficile infection (CDI) can cause life-threating inflammatory responses in the intestinal mucosa and has become the nosocomial infection with the highest medical and economical relevance in Germany. However, our knowledge about host immunity in human CDI is fragmentary, and especially the role of memory effector T cells, like mucosal-associated invariant T (MAIT) cells, remains elusive. MAIT cells are innately pathogen-reactive and restricted by MHC class 1-related protein 1 (MR1), which presents antigenic bacterial metabolites of the riboflavin pathway. Presently the role of MAIT cells

in CDI is unknown but interestingly their blood frequency decreases in age whereby the incidence of CDI increases. Thus, MAIT cells might mediate underscored protection against CDI infection and their functional impairment might increase susceptibility to CDI in elderly.
We performed a comprehensive characterization of the responsiveness of MAIT cells towards *C. difficile*. We confirmed for the first time a functional riboflavin synthesis of *C. difficile* and found fixed bacteria capable of activating primary human MAIT cells in a dose-dependent manner. Moreover, *C. difficile*-activated MAIT cells showed an increased and MR1-dependent expression of CD69, proinflammatory IFNγ, and the lytic granule components granzyme B and perforin. Effector protein expression was accompanied by the release of lytic granules, which, in contrast to other effector functions, was mainly induced by IL-12 and IL-18. Notably, this study revealed hypervirulent *C. difficile* strains to be most competent in provoking MAIT cell responses suggesting MAIT cell activation to be instrumental for the immunopathology observed in *C. difficile*-associated colitis (Bernal et al. 2018, in revision).

Interleukin-8 and the terminal complement pathway identify prosthesis infection in periprosthetic tissue samples ☐

Meinshausen A[1,2], Macor P[4], Pieper D[1,3], Medina E[1,3], Lohmann C H[1,2], Bertrand J[1,2]

[1]ESF International Graduate School on Analysis, Imaging and Modelling of Neuronal and Inflammatory Processes; [2]Department of Orthopaedic Surgery, Otto-von-Guericke University, Magdeburg, Germany; [3]Helmholtz Centre for Infection Research; [4]Department of Life Science, University of Trieste, Italy

Low-grade infections cannot be easily distinguished from aseptic complications frequently leading to false negative diagnoses and late onset of anti-bacterial therapy. Therefore, there is a great need to establish biomarkers for early detection of low-grade infections. In this study, we focused on the investigation of anti-α-defensin, anti-CD68, anti-IL8, anti-C3, anti-C5 and anti-C9 as potential biomarkers for infection in a cohort of hip and knee septic revision cases, taking patient characteristics and comorbidities into account. Here we included 78 patients with septic (35) and aseptic (43) (♀37, ♂42, age 50 – 93 years) revision surgeries of hip and knee. Microbiological diagnostics and next-generation sequencing (NGS) for the identification of bacterial strains was performed. CRP serum levels and leucocyte blood values were evaluated. Patient characteristics, including age, number of prior revision surgeries and comorbidities were recorded. Periprosthetic tissue was stained histologically with Giemsa and Hematoxylin/Eosin and immunhistologically with different

antibodies. The NGS and microbiological diagnostic were concordant in 58,54 % of all cases. Staphylococcus bacterial strains were often false negative in microbiological diagnostic. The CRP values were significantly increased in the septic cohort, but no changes were observed in leucocyte count. Interestingly, we found a strong increase in anti-IL8 (septic: 0.130 ± 0.066, aseptic: 0 ± 0.003 p= 0.0022) staining and the terminal complement system component C9 (septic: 0.099 ± 0.24 aseptic: 0.01 ± 0.049 p= 0.0004) in the septic periprosthetic tissue. Giemsa staining confirmed the chemo attractive capacity of IL-8 by showing more neutrophils in the septic tissue. The predictive value of α-defensin expression was less pronounced (septic: 0.5 ± 0.67 aseptic: 0.103 ± 0.537 p= 0.0867).
We observed a strong predictive value of IL-8 and anti-C9 for tissue infection, suggesting that anti-C9 and ani-IL-8 could be a novel biomarker for the identification of low-grade infections using tissue biopsies.

Role of TRPC channels in temporal bridging and spatial navigation in the hippocampal-entorhinal circuit ☑

Marouf B S[1,2], Yoshida M[2], Sauvage M[3]

[1]ESF International Graduate School on Analysis, Imaging and Modelling of Neuronal and Inflammatory Processes, Medicine Faculty, Institute of Physiology, Otto-von-Guericke University of Magdeburg, [2]German Center for Neurodegenerative Diseases (DZNE), cognitive neurophysiology laboratory,[3]Leibniz-Institute for Neurobiology, Functional Architecture of Memory Department, Magdeburg, Germany

Transient receptor potential (TRP) proteins are cation channels that are responding receptor dependent activation of phospholipase C. Mammalian Transient receptor potential Channels (TRPC) are multigene consisting at least seven proteins (TRPC1-7). TRPCs are known as Ca^{+2} entry channel in the brain. According structural homology, TRPCs can be sorted into three subgroups: TRPC1, TRPC4, and TRPC5 in one subgroup, TRPC3, TRPC6, and TRPC7 in other group and TRPC2 as third subgroup. The prefrontal cortex and hippocampus are two essential key role player of spatial working memory. There are various theories following the neurophysiological basis of transient memory. Most of them can be grouped into two types, which are not mutually exclusive. The first group focuses on enduring spiking within recurrent networks of neurons. The second group highlight non-synaptic mechanisms that are intrinsic to individual neurons. The phenomenon of endogenous

persistent firing is immediate interest in this respect. We hypothesize that cells in hippocampus and entorhinal cortex use persistent firing mechanism by hiring TRPC 4 and TRPC 5 channels to hold received data and further processing in memory and behavior functions. The mechanism of persistent firing in spatial working memory is not clearly understood. The project will verify the role TRPC channels (4 & 5) in persistent firing during a spatial working memory task. Previous studies have revealed the TRPC channel expression in the different parts of the brain. We will use shRNA method and virus injection surgeries on CA 1 region to silence TRPC 4 and 5 channels. Using tetrode recording, CA1 pyramidale cell layer activity will be recorded during behavior tasks (Open field, T-maze with retuning arms, Fear conditioning and Trace fear conditioning). Statistical analysis and spike sorting will be done using SPSS, Matlab and TINT spike sorting software to analyze recorded electrophysiology and behavioural test results

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Metagenomic Survey of Microbiota Function in Laryngeal Tissues in the development of Dysplasia and Carcinoma ☐

Wang Y[1, 2], Arens C[1,2], Naumann M[1,3]

[1]*ESF International Graduate School on Analysis, Imaging and Modelling of Neuronal and Inflammatory Processes;* [2]*Clinic for Ear, Nose and Throat Medicine, Otto-von-Guericke University Magdeburg, Germany;* [3]*Institute of Experimental Internal Medicine, Medical Faculty, Otto-von-Guericke-University Magdeburg, Germany*

Head and neck squamous cell carcinomas (HNSCC) as frequently occurring cancers have over 500,000 new cases diagnosed worldwide each year. Laryngeal carcinoma (157,000 new cases every year) is one of the most common malignancies of the head and neck, and the most frequent histological type of it is squamous cell carcinoma, accounting for 98% of cases. The sex ratio (7:1) is greater than for any other cancers, it is a rare cancer in women with only 19,000 new cases worldwide. Until now, the cause of laryngeal cancer is not very clear. The microbial community living on the mucosa of the larynx builds a stable microenvironment and it may affect the health of human larynx. There are much evidence about the role of microorganisms in carcinogenesis, including *Helicobacter pylori* and gastric cancer, Epstein-Barr virus and nasopharyngeal cancer, *Fusobacterium nucleatum* and colorectal cancer. However, the association between microbiota and the development of laryngeal cancer remains uncertain. At present, microbial research on tissues from human body mainly uses mark gene analysis (16s rRNA), and although the metagenomic analysis can directly

conclude the relative abundance of microbial functional genes, it is rarely used in clinical tissue specimens. Here, in the present study, the use of metagenomic sequencing will be explored to solve microbiota composition in laryngeal tissues and its impact on the development of dysplasia and carcinoma with control subjects from vocal cord polyps. These studies can help to understand the role of microbiota in laryngeal carcinogenesis, explore new molecular markers, and develop personalized therapies.

Effort processing in the substantia nigra – intracranial recordings from patients suffering from Parkinson's disease ☐

Repplinger S[1,2,3], Heinze HJ[1,2,3], Zähle T[1,2]

[1]ESF International Graduate School on Analysis, Imaging and Modelling of Neuronal and Inflammatory Processes; [2]Departments of Neurology and Stereotactic Neurosurgery, Otto-von-Guericke University, Magdeburg, Germany; [3]Department of Behavioral Neurology, Leibniz Institute for Neurobiology, Magdeburg, Germany

Expectation of reward leads to the release of dopamine in midbrain structures. This is an accepted finding in neuroscientific research. Processing an upcoming reward in the purpose of decision-making, every intentional action presupposes an implicit weighing of benefits against costs. These cost-benefit calculations are attributed to substructures of the basal ganglia, namely the substantia nigra (SN) and

ventral tegmental area (VTA). Previous studies highlighted the functional role of these regions using noninvasive imaging in humans, as well as single cell recordings in animals. Increasing the expected reward led to an increase of BOLD response indicating that the task effort acts as a modulatory factor. Higher effort resulted in attenuation of the activation. The aforementioned cost-benefit calculation is the proposed mechanism explaining this finding.

Due to the indirect nature of the BOLD response in fMRI recordings, there is still a lack in direct evidence from humans. This study shall bridge the gap between findings from invasive electrophysiology in animals and noninvasive imaging in humans. For this purpose, we intraoperatively record electrophysiological data in patients suffering from Parkinson's disease. Undergoing surgery for Deep Brain Stimulation, microelectrodes are implanted into the patients' SN. In a two alternative forced-choice task, patients discriminate shapes of varying complexity reflecting two levels of effort. We expect a modulation of the intracranial EEG signal at about 200 and 800 milliseconds time-locked to cue occurrence, indicating the upcoming effort level. These data will provide direct evidence for the effortsensitivity of dopamine neurons in the absence of reward in humans.

Visual search and attention guidance in patients with macular degeneration ☐

Sharavanan G[1, 2], Hanke M[1, 2], and Pollmann S[1, 2]

[1]ESF International Graduate School on Analysis, Imaging and Modeling of Neuronal and Inflammatory Processes, [2]Department of Experimental Psychology, Institute of Psychology, and Center for Behavioral Brain Sciences, Otto-von-Guericke University, Magdeburg, Germany.

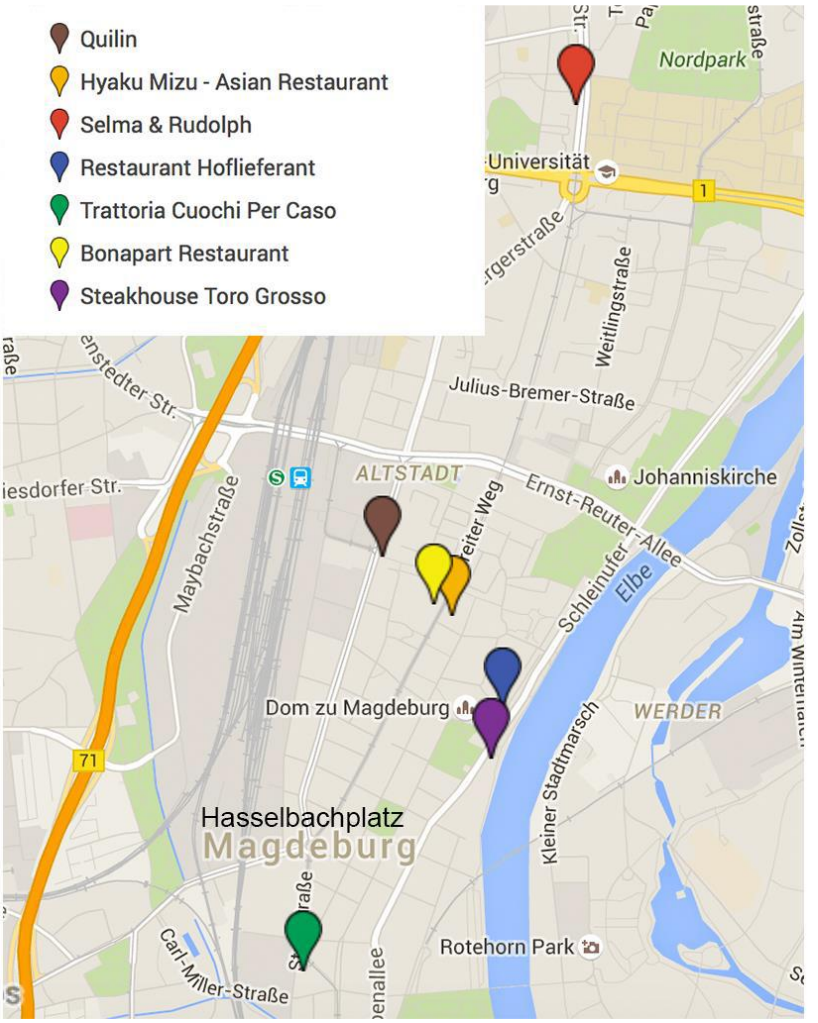
Age-related macular degeneration (AMD), affecting the central vision, is the major cause for reduced visual acuity in the geriatric population. To compensate for the functional loss partially, most patients with AMD adopt an eccentric preferred retinal location (PRL). Studies on contextual cuing in visual search on AMD patients have shown poor performance that is mainly due to lack of memory-guided search with inefficient saccadic exploration. On the other hand, saccades redirected to PRL, saccadic re-reference (SR), develops slowly after months of oculomotor adaptation following PRL development. Recent studies with central vision loss (CVL) simulation have shown different paradigms where SR can be induced successfully within hours. One of the main aims of this study is to develop a SR training method that is efficient, stable, and quick on AMD subjects and normals with simulated CVL. In addition, we also wish to understand the effects of SR training on memory-guided search in the contextual cueing paradigm. Lastly, we plan to investigate any transfer effects of SR training on other visual tasks like reading. For SR training, we plan to use a visual foraging task with multiple targets among a group of distractors; subjects will be trained to fixate targets with a pre-determined perifoveal location using a gaze-contingent eye tracking system. The position and variability of

the first saccade landing location after target presentation will be measured and analyzed. Search time, number of fixations made while searching a target, reading speed, and fixation stability evaluation estimated from bivariate contour ellipse area (BCEA) will be measured and compared. The results of this study may lead to a training program for patients with AMD.

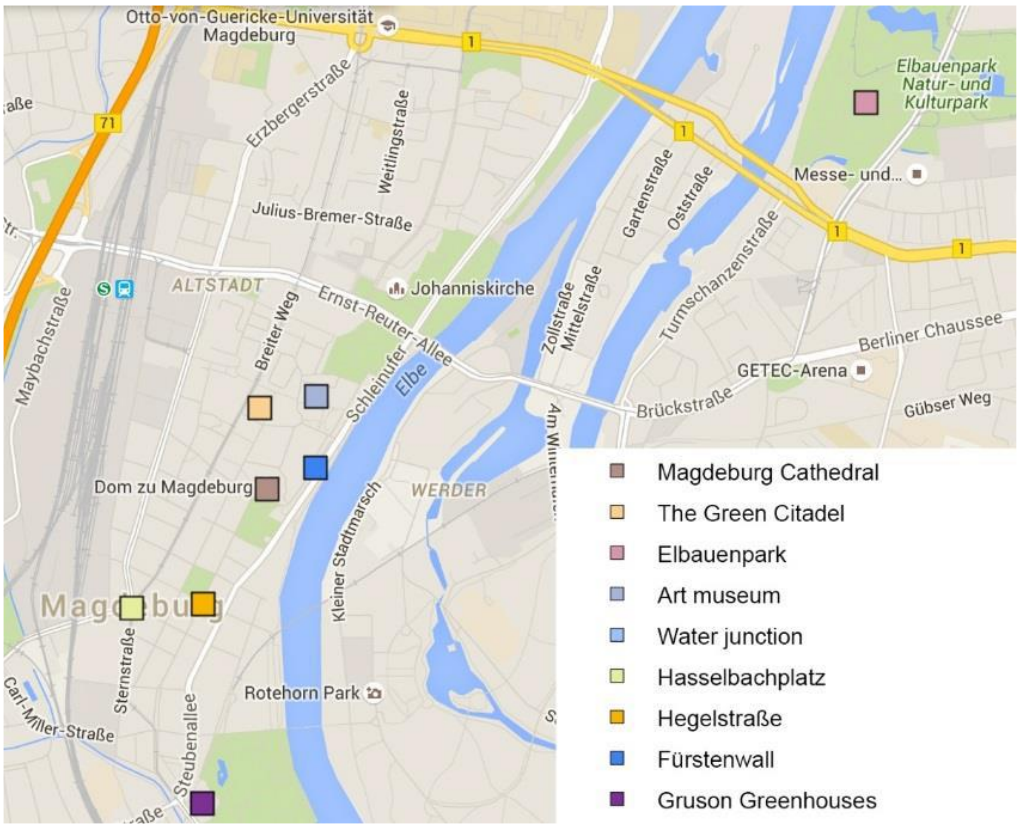
Recreational Information

Restaurants

The district around Hasselbachplatz is home to many pubs, bars, and restaurants and is well worth to visit, especially in the evening.



Sightseeing



The water junction cannot be reached via public transport. Here is the phone number of a local cab company: 0049 391 737373


189
2
Lostau
Barleben
Gerwisch
71
189
Biederitz
Woltersdorf
1
1
Königsborn
184
Gübs
184

Downtown/Oldtown

The Green Citadel of Magdeburg

One of Magdeburg's most eye-catching attractions for visitors is also one of the last architectural masterpieces designed by the artist Friedensreich Hundertwasser. The Hundertwasser Building is located among a mixture of Baroque facades and examples of modern design.

Magdeburg Cathedral

Magdeburg Cathedral is the first Gothic-style cathedral to be constructed on German soil, one of the largest church buildings in Germany and the most famous attraction in Magdeburg, the capital city of the German federal state of Saxony-Anhalt.

Art Museum in the Monastery of Our Lady

The Art Museum in the Monastery of Our Lady is the most important venue for contemporary art and sculpture in the German federal state of Saxony-Anhalt and is one of the most popular tourist attractions in the region.

Fürstenwall

The Fürstenwall area dates back to the Middle Ages and contains city fortifications facing the river Elbe and the two preserved fortified towers. Built in 1725, this was the first public promenade in Germany. The neighbouring Bailiwick Garden (German: Möllenvogteigarten) is the oldest preserved garden design in the city of Magdeburg.

Hegelstraße

The Hegelstraße begins at the Cathedral and runs in a southerly direction. The popular boulevard was built between 1880 and 1920, during the Gründerzeit era, and is lined with magnificent representative buildings.

Hasselbachplatz

At Hasselbachplatz, which is named after a former major of Magdeburg (1809-1882), you can marvel at the city's most magnificent Gründerzeit-style facades. This district is home to many pubs, bars and restaurants and is well worth a visit, especially in the evening.

Close by…

Grusons Greenhouses

The «Gruson-Gewächshäuser» are a collection of greenhouses that are home to a traditional botanical garden featuring the exotic collection of plants kept by the industrialist Herrmann Gruson from the German city of Magdeburg.

The Elbauenpark

With its unique «Seebühne» Lake Stage and the Millennium Tower, the world's tallest wooden construction of its kind, the Elbauenpark in the German city of Magdeburg is well worth a visit 365 days a year.

Waterway Junction

The water-saving lock Rothensee and the longest canal bridge in Europe (918 m) which spans the river Elbe, the double ship lift Hohenwarthe and the connecting canals are all part of a gigantic building project to connect the waterways of Hannover, Magdeburg and Berlin.

List of Delegates

Agostino, Camila Silveira (OVGU, FME, IPSY)	*camila.agostino@ovgu.de*
Arens, Christoph (OVGU, FME, KHNO)	*arens.christoph@med.ovgu.de*
Barbazzeni, Beatrice (OVGU, FME, KNEU)	*beatrice.barbazzeni@med.ovgu.de*
Bernal, Isabel (OVGU, FME, IMMB, HZI)	*isabel.bernal@helmholtz-hzi.de*
Bertrand, Jessica (OVGU, FME, KORT)	*jessica.bertrand@med.ovgu.de*
Bhattacharjee, Rituparna (OVGU, FME, IMMB)	*rituparna.bhattacharjee@med.ovgu.de*
Boehme, Julia (OVGU, FME, IMMB, HZI)	*julia.boehme@helmholtz-hzi.de*
Braun, Jochen (OVGU, FNW, IBIO)	*jochen.braun@ovgu.de*
Bruder, Dunja (OVGU, FME, IMMB, HZI)	*dunja.bruder@med.ovgu.de*
Budinger, Eike (LIN)	*eike.budinger@lin-magdeburg.de*
Brunner-Weinzierl, Monika (OVGU, FME, KPAE)	*monika.brunner-weinzierl@med.ovgu.de*
Canbay, Ali (OVGU, FME, KGHI)	*ali.canbay@med.ovgu.de*

Cangalaya Lira, Carla Marcia (OVGU, FME, IBZ, DZNE)	*carla.cangalaya@ovgu.de*
Dityatev, Alexander (DZNE)	*alexander.dityatev@dzne.de*
Dunay, Ildiko (OVGU, FME, IIN)	*ildiko.dunay@med.ovgu.de*
Düzel, Emrah (OVGU, FME, IKND, DZNE)	*emrah.duezel.dzne.de*
Edelmann, Elke (OVGU, FME, IPHY)	*elke.edelmann@med.ovgu.de*
Färber, Jacqueline (OVGU, FME, IMMB)	*jacqueline.faerber@med.ovgu.de*
Fischer, Klaus-Dieter (OVGU, FME, IBZ)	*klaus.fischer@med.ovgu.de*
Fischer, Thomas (OVGU, FME, KHAE)	*thomas.fischer@med.ovgu.de*
French, Timothy (OVGU, FME, IPSY)	*timothy.french@med.ovgu.de*
Ganesan, Sharavanan (OVGU, FME, IPSY)	*sharavanan.ganesan@ovgu.de*
Goldschmidt, Jürgen (LIN)	*jurgen.goldschmidt@lin-magdeburg.de*

Gopala, Nishanth (OVGU, FME, IMMB)	*nishanth.gopala@med.ovgu.de*
Gorny, Xenia (OVGU, FME, KNEP)	*xenia.gorny@med.ovgu.de*
Gundelfinger, Eckart (LIN)	*eckart.Gundelfinger@lin-magdeburg.de*
Hanke, Michael (OVGU, FNW, IPSY)	*michael.hanke@gmail.com*
Hedtmann, Christiane (OVGU, FME, IPHY, ABINEP)	*christiane.hedtmann@med.ovgu.de*
Heinze, Hans-Jochen (OVGU, FME, KNEU)	*birgit.boehme@med.ovgu.de (Sekretariat)*
Hinrichs, Hermann (OVGU, FME, KNEU)	*hermann.hinrichs@med.ovgu.de*
Jänsch, Lothar (HZI)	*lothar.jaensch@helmholtz-hzi.de*
Jocham, Gerhard (OVGU, FWW, CNL)	*gerhard.jocham@ovgu.de*
Kakaei, Ehsan (OVGU, FNW, IBIO)	*ehsan.kakaei@ovgu.de*
Khodaie, Babak (OVGU, FME, IPHY)	*babak.khodaie@med.ovgu.de*
Kröger, Andrea (OVGU, FME, IMMB)	*andrea.kroeger@med.ovgu.de*

Lessmann, Volkmar (OVGU, FME, IPHY, ABINEP)	*volkmar.lessmann@med.ovgu.de*
Lohmann, Christoph (OVGU, FME, KORT)	*christoph.lohmann@med.ovgu.de*
Malci, Ayse (LIN)	*ayse.malci@lin-magdeburg.de*
Marouf, Babak Saber (DZNE)	*babak_sm@msn.com*
Medina, Eva (HZI)	*eva.medina@helmholtz-hzi.de*
Meishausen, Ann-Kathrin (OVGU, FME, KORT)	*ann-kathrin.meishausen@med.ovgu.de*
Mertens, Peter (OVGU,FME, KNEP)	*mertens.peter@med.ovgu.de*
Müller, Andreas (OVGU, FME, IMKI)	*andreas.mueller@med.ovgu.de*
Munsch, Thomas (OVGU, FME, IPHY)	*thomas.munsch@med.ovgu.de*
Naumann, Michael (OVGU, FME, IEIM)	*naumann@med.ovgu.de*
Noesselt, Tömme (OVGU, FNW, IfP)	*toemme.noesselt@med.ovgu.de*
Ohl, Frank	*frank.Ohl@lin-magdeburg.de*

(OVGU, FNW, IBIO, LIN)	
Osbelt, Lisa (OVGU, FME, IMMB, HZI)	*lisa.osbelt@helmholtz-hzi.de*
Pausder, Alexander (OVGU, FME, IMMB, HZI)	*alexander.pausder@helmholtz-hzi.de*
Pieper, Dietmar (HZI)	*pieper.dietmar@helmholtz-hzi.de*
Pollali, Evangelia (OVGU, FNW, IBIO)	*polleva@ymail.com*
Pollmann, Stefan (OVGU, FNW, IfP)	*stefan.pollmann@ovgu.de*
Repplinger, Stefan (OVGU, FME, KNEU)	*stefan.repplinger@med.ovgu.de*
Rogge, Julia (OVGU, FNW, IfP)	*jrogge@ovgu.de*
Rössner, Albert (OVGU, FME, IPA)	*albert.roessner@med.ovgu.de*
Ryll, Anke (OVGU, ABINEP)	*anke.ryll@ovgu.de*
Sauvage, Magdalena (LIN)	*magdalena.Sauvage@lin-magdeburg.de*

Schalk, Enrico (OVGU, FME, KHAE)	*enrico.schalk@med.ovgu.de*
Schlüter, Dirk (OVGU, FME, IMMB)	*dirk.schlueter@med.ovgu.de*
Schmitz, Ingo (OVGU, FME, IMKI, HZI)	*igno.schmitz@helmholtz-hzi.de*
Schott, Björn (LIN)	*bschott@neuro2.med.uni-magdeburg.de*
Schreiber, Jens (OVGU, FME, KPNE)	*jens.schreiber@med.ovgu.de*
Schreier, Sarah (OVGU, FME, IMMB, HZI)	*sarah.schreier@med.ovgu.de*
Schulz, Andreas (LIN)	*andreas.Schultz@lin-magdeburg.de*
Seidenbecher, Constanze (LIN, ABINEP)	*seidenc@lin-magdeburg.de*
Shah, Aneri Tusharbhai (OVGU, FME, KNEP)	*aneri.shah@med.ovgu.de*
Speck, Oliver (OVGU, FNW, BMMR)	*oliver.speck@ovgu.de*
Stoyanov, Stoyan (DZNE)	*stoyan.stoyanov@dzne.de*

Strackeljan, Jens Rektorand	*rektor@ovgu.de*
Strowig, Till (HZI)	*till.strowig@helmholtz-hzi.de*
Stork, Oliver (OVGU, FNW, IBIO)	*oliver.stork@ovgu.de*
Ullsperger, Markus (OVGU, FNW, IfP)	*markus.ullsperger@ovgu.de*
Viswanathan, Vivekanan-dhan (LIN)	*viveksugan@gmail.com*
Wang, Xu (OVGU, FME, IMMB)	*xu.wang@med.ovgu.de*
Yoshida, Motoharu (DZNE, LIN)	*motoharu.yoshida@dzne.de* *motoharu.yoshida@lin-magdeburg.de*
Zähle, Tino (OVGU, FME, KNEU)	*tino.zaehle@ovgu.de*
Zenker, Martin (OVGU, FME, IHG)	*martin.zenker@med.ovgu.de*

Notes